method. Hence, it represents a valuable tool for the clinician to assess the trough levels during ADA treatment follow up at the point of care.

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Disclosure of Interest: F. Bantleon Employee of: Employee of BÜHLMANN Laboratories AG, S. Kräuchi Employee of: Employee of BÜHLMANN Laboratories AG, T. Schuster Employee of: Employee of BÜHLMANN Laboratories AG, M. Schneider Employee of: Employee of BÜHLMANN Laboratories AG, J. Weber Employee of: Employee of BÜHLMANN Laboratories AG

DOI: 10.1136/annrheumdis-2017-eular.4981

AB1167 AUTOREACTIVE T CELLS TO CITRULLINATED HSP90 IN INTERSTITIAL LUNG DISEASE IN RHEUMATOID ARTHRITIS

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Background: Previous studies have indicated that anti-citrullinated protein antibody (ACPAs) titers are associated with the presence and severity of interstitial lung disease (ILD) in RA. The lung might be a site of initiation of immunity to citrullinated proteins. Previous studying anti-citrullinated HSP90 (citHSP90β) profiles between bronchial veolarlavage fluid (BALF) and serum indicated the lung plays a direct role in shaping the immune repertoire of RA-ILD.
Objectives: Whether citHSP90β contributes to the initiation or perpetuation of autoimmune interstitial lung abnormalities (ILA) in RA remains to be clarified. To address this issue we investigated the spontaneous T cell responses to the putative autoantigen citrullinated HSP90β in different stages of RA-ILD.
Methods: In RA-no ILD (n=19), indeterminate ILD (ILA1) (n=24), subclinical RA-ILD (ILA 2) (n=20), clinical RA-ILD (ILA3) (n=4) and other connective tissue disease associated ILD (CTD-ILD) (n=14) patients. Cultures derived from whole blood were individually stimulated with HSP90β, citHSP90β, citrullinated BSA, or no antigen. The concentration of 13 cytokines and chemokines in the plasma supernatant were then measured using Luminex xMAP technology.
Results: CitHSP90β induced significantly higher levels of IFN-γ levels in RA-ILD (ILA=2+3) groups compared to the RA-no ILD group (p=0.002), but did not stimulate the production of other cytokines (p>0.05). Furthermore, citHSP90β did not stimulate the production of IFN-γ or other cytokines stimulated those individuals with non-RA CTD-ILD (p=0.1039, IFN-g).
Conclusions: The production of IFN-γ by T cells stimulated with citHSP90b demonstrates a bias toward TH1 immune responses that are likely involved in the pathogenesis of RA-ILD. The presence of autoreactive Th1-like cells in RA patients in conjunction with citrullinated autoantigens may indicate the involvement of this autoantigen in the pathogenesis of RA-ILD. Early targeting of the immune reactions in the lung might therefore be a new approach to modulate disease.

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Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.2596

AB1168 TOUCH STUDY: TECHNOLOGY AND OUTCOMES USED IN CLINIC IN A DAY HOSPITAL

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Background: Patient reported outcomes PRO are a key element in the global evaluation of patients, especially those followed in a day hospital. The use of touchscreen computers is one of the new features in the day hospital of Instituto Português de Reumatologia.
Objectives: to evaluate the transition from paper to touchscreen computers technology of the PRO in use in Reuma.pt
Methods: We considered a step up model of comparison with 2 months intervals one before the use of the touchscreen computers, one two months after the introduction of touchscreen computers and a third after an intermediate evaluation (comparison between interval 0 and 1) of the results.A specific formation to physicians and nurses to be aware of missing data from non-total completion of the questionnaires was introduced between the first and second evaluation. The percentage of questionnaires totally completed by number of patients were obtained for every period and diagnosis
Results: 631 day hospital appointments were evaluated according to diagnosis and interval and the percentage was obtained (Table 1)

Table 1. Results comparing questionnaires by diagnosis and intervals

	Paper interval 0 (Sept –Nov 15)			Interval 1 (Nov 15- Jan 16)			Interval 2 (Jan – Mar 16)		
AS	N	N Quest.	Pct.	N	N Quest.	Pct.	N	N Quest.	Pct.
BASDAI	95	95	100,00%	92	87	94,57%	93	92	98,92%
BASFI	95	94	98,95%	92	89	96,74%	93	92	98,92%
EQ5D	95	91	95,79%	92	85	92,39%	93	88	94,62%
AsQol	95	88	92,63%	92	83	90,22%	93	85	91,40%
SF-36	95	80	84,21%	92	72	78,26%	93	77	82,80%
HADS	95	27	28,42%	92	87	94,57%	93	91	97,85%
FACIT	95	93	97,89%	92	91	98,91%	93	92	98,92%
RA									
HAQ	112	111	99,11%	124	124	100,00%	115	114	99,13%
SF-36	112	96	85,71%	124	101	81,45%	115	98	85,22%
HADS	112	9	8,04%	124	111	89,52%	115	114	99,13%
FACIT	112	108	96,43%	124	122	98,39%	115	114	99,13%
EQ5D	112	105	93,75%	124	119	95,97%	115	113	98,26%

Only HADS had a significative (p<0.000) improvement for every disease, with the use of the touchscreen computers from interval 1 to 2. On our intermediate evaluation comparing paper to tablet we saw a lower percentage of questionnaires fully completed (although not statistical significative) and a formal awareness formation addressing the causes was made with all the physicians and nurses of the day hospital. The PRO from Reuma.pt was not developed for tablets and some issues regarding missing data associated with that was found
Conclusions: The use of technology can contribute for better data in Reuma.pt and other national registries by saving time (medical and nurse) for clinical evaluation, by integrating patients in their evaluations and by cost reduction, and carbon footprint. Issues regarding the adaptability of software to tablet technology have to be addressed to insure an overall improvement.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.6897

AB1169 COMPENSATORY INCREASE IN FCΓRIIB EXPRESSION ON B CELLS IN PATIENTS WITH SYSTEMIC SCLEROSIS

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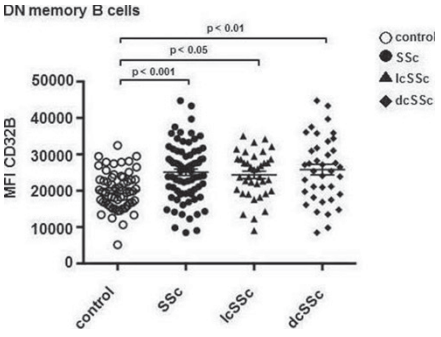
Background: Systemic sclerosis (SSc) is a systemic autoimmune disease that is characterized by excessive fibrosis and microvascular damage. The contribution of B cells to the pathology of SSc has been mainly recognized as polyclonal B cell activation and autoantibody production. The receptor for the Fc region of IgG (FcγRs) is member of the Ig superfamily, the members of which modulate the activation and inhibition of immune responses. FcγRIIB is the only FcγR that has an inhibitory function. Previous studies revealed that a decrease in the expression of FcγRIIB on B cells induced excessive immune responses and resulted in the development of autoimmunity. However, the expression levels of FcγRIIB on B cells in SSc currently remain unknown.
Objectives: We aimed to clarify how the abnormal activation of B cells involves inhibitory FcγRIIB on B cells in SSc patients.
Methods: Blood samples were collected from 76 SSc patients (38 limited cutaneous SSc [lcSSc], 38 diffuse cutaneous SSc [dcSSc]) and 59 healthy subjects. We evaluated the expression of FcγRIIB, CD80, CD86, and CD95 on B cell subsets. B cells were classified into five subsets depending on their surface molecular expression by flow cytometry: naïve B cells ($CD19^+IgD^+CD27^-$), preswitched memory B cells ($CD19^+IgD^+CD27^+$), double negative (DN) memory B cells ($CD19^+IgD^-CD27^-$), switched memory B cells ($CD19^+IgD^-CD27^{mid}$), and plasmablasts ($CD19^+IgD^-CD27^{high}$). The mRNA expression of FcγRIIB was measured using real-time PCR. We examined the relationship between FcγRIIB expression and clinical features.
Results: The expression of FcγRIIB on SSc naïve B cells and DN memory B cells was significantly stronger than that on the B cells of healthy subjects (Figure 1) (p<0.05 and p<0.001, respectively). FcγRIIB mRNA expression on SSc B cells was also significantly stronger than that on the B cells of healthy subjects (p<0.01). The expression of CD80, CD86, and CD95, activation markers on B cells, was stronger in all 5 B cell subsets, except for CD80 in switched memory B cells and plasmablasts. Patients with the stronger expression of FcγRIIB on DN memory B cells more frequently had interstitial lung disease than those with normal levels (p<0.05). Cyclophosphamide pulse therapy significantly reduced the expression of FcγRIIB on preswitched memory B cells and switched memory B cells (p<0.05 and p<0.05, respectively).



Conclusions: Our results suggest that SSc B cells exhibit compensatory increases in the expression of FcγRIIB in order to suppress the abnormal activation of B cells, and the expression of FcγRIIB may be an indicator of the clinical severity of SSc.
Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.2064

AB1170 PREVALENCE OF ANTI-CARP ANTIBODIES IN PATIENTS WITH PRIMARY SJÖGREN'S SYNDROME: ASSOCIATION WITH CLINICAL, SEROLOGICAL AND HISTOLOGICAL ASPECTS

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Background: The presence of antibodies against carbamylated peptides (anti-CarP) has been associated with increased disease activity and severe joint damage in rheumatoid arthritis. Our group has demonstrated their presence also in other inflammatory conditions with a high prevalence in Sjogren's Syndrome (SS) (14/45, 31.1%)[1]. There is only one study up to now investigating anti-CarP in SS demonstrating a prevalence of these antibodies in 27% of cases and an association with the presence of germinal centres (GCs)[2]. Thus, these antibodies have been proposed as a new tool for identifying SS patients with a more aggressive disease.
Objectives: To confirm the presence of anti-CarP antibodies in a large mono-centric cohort of patients with SS and investigate their association with clinical, serological and histological features.
Methods: Serum samples from consecutive patients with SS (AECC criteria) were collected and stored at -20°. Anti-CarP antibodies were detected by a modified solid-phase "home-made" ELISA[1]. The mean +3 times SD was used as cut-off. Minor paraffin embedded salivary glands were stained by H&E and IHC using lymphocytes T and B markers [anti-CD3, anti-CD20 (DAKO)]. GCs presence was defined by H&E and confirmed by identification of follicular dendritic cells (anti-CD21, DAKO). Images were analysed as follows: focus score (FS) calculation, mean foci area, presence of segregated foci (SF), GCs and lymphoepithelial lesions (LELs).
Results: Clinical and laboratory features of SS patients are shown in table. Serum anti-CarP were detected in 30/104 patients (28.8%) without association with any clinical or serological feature (Fisher's exact test). Positive patients were more likely to present SF (P=0.024). No association was found with the presence of GCs or LELs. Anti-CarP titre correlated with the FS (P=0.045, r=0.304), the number of foci (P=0.008, r=0.347), mean foci area (P=0.028, r=) and the percentage of SF (P=0.046, r=0.331) (Spearman's test). Prevalence of anti-CarP was higher in patients with arthritis [7/15 (46.6%)] than those without ([23/89 (25.8%)] (p>0.05).

Clinical features	Number (%)
Sex (M/F)	5/99
Age at diagnosis (mean±SD, years)	53±11.7
Xerophthalmia	93/104 (89.4)
Xerostomia	97/104 (93.2)
Glandular swelling	31/104 (29.8)
Arthritis	15/104 (14.4)
Lymphoma	3/104 (2.8)
Laboratory features	
ANA	88/104 (84.6)
Anti-Ro/SSA	54/104 (51.9)
Anti-La/SSB	42/104 (40.3)
Hypergammaglobulinemia	28/104 (26.9)
Rheumatoid factor	38/104 (36.5)
Leucopenia	21/104 (20.1)
Hypocomplementemia	9/104 (8.6)
Monoclonal component	8/104 (7.6)
Cryoglobulinemia	5/104 (4.8)

Mean anti-CarP titre was higher in patients with arthritis compared to those without (322.3±173.8 aU/ml vs 279.5±171.1, P=0.004, respectively).
Conclusions: This is the largest cohort of SS patients screened for anti-CarP so far. Our results show a prevalence of anti-Carp antibodies in agreement with the literature; however, no association was found with any clinical or serological aspect. Anti-CarP do not seem to be associated with histological features predictive of lymphoma, i.e. CGs and LELs. Nonetheless, considering the titre correlation with the FS, mean foci area and percentage of segregation, higher serum levels of anti-CarP may reflect a severe tissue inflammation more prone to form organized infiltrates. This finding, in association with the evidence of higher levels in patients with arthritis, may support the idea that these antibodies are useful to measure the severity of systemic inflammation.
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Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.5774

AB1171 ARTICULAR AND EXTRA-ARTICULAR DAMAGE INDEX ASSESSMENT IN JUVENILE IDIOPATHIC ARTHRITIS PATIENTS

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Background: Juvenile Idiopathic Arthritis is a broad term used to describe several different forms of chronic arthritis in children. Variable assessment tools can be used for assessment of JIA disease activity. JADI is composed of 2 parts (Viola et al., 2005).
Objectives: Assessment of the articular and extraarticular damage index in juvenile idiopathic arthritis patients.
Methods: This study was carried out on 60 JIA patients. Patients who had inflamed synovia related to trauma or malignant disease, septic arthritis, bone diseases, as dysplasia or osteomyelitis were excluded. *Clinical assessment:* sex, age at disease onset, JIA category, educational level, loss of school years and previous use of systemic corticosteroid and second-line drug therapies. *Local examination:* Number of swollen joints, joints with pain on movement/tenderness, joints with limited range of motion, joints with active arthritis were recorded for every patient. *Disease activity:* was measured by using the (JADAS-27). *Functional ability:* was assessed by (CHAQ). *Laboratory assessment:* included ESR and CRP. *Radiographic assessment:* scored according to the adapted Sharp/van der Heijde score. *Damage assessment:* was assessed using the Juvenile Arthritis Damage Index.The (JADI-A) and (JADI-E).
Results: *Patient characteristics:* 6 had systemic onset, 29 had polyarthritis, 14 extended oligoarthritis, 11 had persistant oligoarthritis and none of them had psoriatic arthritis. 38 females and 22 males (56.7%) percent of patients lost some years of education ranging from 0–3 years. Patients in remession were very few 5 patients only. According to the C-HAQ score (13.3%) of patients had no disability (11.7%) mild disability (41.6%) moderate disability and (33.3%)severe disability. 60% patients had articular damage and 35% patients had extraarticular damage. The wrist was the most frequently damaged joint.The growth failure, pubertal delay and leg length discrepancy were the most frequently reported extraarticular items. *Correlation of JADI with other disease variables:* showed that JADI-A is correlated with physician's global assessment, CHAQ, radiological damage.
Conclusions: Ours is the first study that has used JADI to assess outcome in patients with JIA in Egypt. JADI has a good correlation with traditional outcome measures in JIA and may be a good tool to be used in clinical practice and is likely to increase current understanding of the natural history of the disease.
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Acknowledgements: First of all, thanks to Gracious Allah. I would like to express my profound gratitude and appreciation to Prof. Dr. Enass Abdel Kader Elewa and to Prof. Dr. Mahmoud Moustafa Ashour for support and supervision.
Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.6707